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Answers

Marine heatwaves: when you can't cool down in the sea

Answers to practice exam questions on pp. 10–15

Martin Rowland

1

a Caused by/resulting from human activity

b Increase in release of greenhouse gases/of carbon dioxide/of methane/of nitrous oxide

From industry/from agriculture

(Their) molecules absorb electromagnetic radiation/light and re-emit it as heat.

2 Reduces upward diffusion of nutrients/minerals/ions (from benthic layer).

Plus 2 marks for any two of the following:

(So) fewer magnesium ions to make chlorophyll.

(So) fewer nitrate ions/phosphate ions to make amino acids/DNA/RNA.

(So) fewer sulfate ions to make (sulfur-containing) amino acids/cysteine/methionine.

Personalised nutrition: are fruit flies the key?

Answers to practice exam questions on pp. 22–24

Martin Rowland

1 Fruit flies and humans share the insulin/insulin-like growth factor signalling pathway.

CG4607 gene in fruit flies encodes a homologue of the human glucose transporter/is homologue of human gene *SLC2A8*.

If neither of the above given, allow 1 mark for:

Fruit flies share some metabolism-controlling genes with humans.

Fruit fly life span is (much) shorter than human life span, allowing life-long investigations.

Diets of fruit flies can be easily manipulated.

Large number of (genetic) strains of fruit fly allow investigation of effect of genotype.

2 One cause of type 2 diabetes is obesity/overweight/high BMI/high waist-to-hip ratio.

A change of diet can result in weight loss, which reduces insulin resistance of cells.

Type 1 diabetes is caused by an autoimmune response against insulin-producing cells/beta cells.

Insulin-producing cells/beta cells in pancreas cannot be regenerated by change in diet.

Evaluating experiments: where are all the Christmas turkeys?

Suggested answers to questions on pp. 38–41

Kevin O'Dell

Question 1

Island populations are likely to be small and inbred, so from a genetic perspective they are likely to have relatively little variation at a DNA level. Therefore, within a small, inbred population, most or all individuals will have a genetically similar immune system, so will all be similarly resistant or susceptible to a new infection. In the larger and genetically more variable mainland populations, individuals will have genetically different immune systems, and are therefore extremely unlikely all to be highly susceptible to the same infection.

Question 2

Mothers would not be able to pass on a Z-linked gene or mutation to their daughters. This is equivalent (but the other way around) to that seen in mammals, where fathers are not able to pass on a X-linked gene or mutation to their sons.

Question 3

If the sprout phenotype is caused by a rare autosomal recessive mutation at a single gene, then:

- It would be rare in the population, as affected individuals would have to be homozygous for the rare recessive mutation.

- It would be more likely to appear in small, isolated populations, as there would be a greater probability of a heterozygote meeting and then mating with another heterozygote that shares ancestry.
- It would be just as likely to occur in males and females since it is not sex-linked.
- It may disappear for several generations and reappear later as it could survive in a heterozygous state during those generations.

Question 4

If you were able to capture wild Christmas turkeys, you could set up a true-breeding population of Christmas turkeys with sprouts (according to the hypothesis that these would be homozygous for the recessive sprout allele, –/–) and another true-breeding population from isolated parts of the island with no recorded examples of Christmas turkeys with sprouts (which would be homozygous for the dominant wild-type allele, +/+). If you were to cross the two populations together then, for the theory to be correct, all F1 birds would be wild-type (sproutless, +/–). If you were to cross the F1 birds together, the F2 birds would be in a ratio of three wild-type sproutless (+/+, +/–, –/+) Christmas turkeys to one with sprouts (–/–).

Question 5

The key here is to design experiments that test sexual preference in both males and females. These could be experiments that test female choice (where a female is given the opportunity to mate with two males, one with sprouts and one without) and male choice (where a male is given the opportunity to mate with two females, one with sprouts and one without).

In each case you would record which male/female mated first (with or without sprouts). If the mating frequencies were more-or-less equal, then you would conclude that there is no evidence for sexual selection. However, if turkeys with or without sprouts mated significantly more frequently, then you would conclude that sexual selection may be occurring. Sexual selection could occur in one or both sexes.

Question 6

Professor Singer and her team could use a polymerase chain reaction (PCR) test to determine whether Christmas turkeys were infected with Noelvirus. In this case they would use PCR primers that bind to the DNA sequence of the Noelvirus strain brought to Christmas Island by the three donkeys, and amplify a short sequence of Noelvirus DNA. This is how testing for Covid-19 was conducted during the recent pandemic.

Question 7

You would use a chi-squared test. This is used to determine whether there is a statistically significant difference between the observed and expected frequencies in one or more categories of a contingency table. In this case the data presented in Table 1 are presented in a 2 × 2 contingency table, and so are appropriate for a chi-squared test.

Question 8

Even without doing a chi-squared test it is quite clear that far more birds infected with Noelvirus survive if they have sprouts than if they do not have sprouts. Clearly some Christmas turkeys with

sprouts die after infection with Noelvirus and some Christmas turkeys with sprouts do not die after infection with Noelvirus.

Professor Singer's hypothesis would work if the Noelvirus-resistance mutation arose in a gene that mapped close to the rare *sprout* allele at the *sprout* gene. It follows that infected birds carrying the Noelvirus-resistance mutation would survive and as the Noelvirus-resistance allele is closely linked with the *sprout* allele, most of the survivors would also have the sprout phenotype. It follows that the *sprout* allele would effectively increase in frequency in the population because it is coinherited with the Noelvirus-resistance allele.

Question 9

We know that the *sprout* gene and Noelvirus-resistance are closely linked on the same chromosome. Therefore, we know the approximate chromosomal position of the Noelvirus-resistance gene, but this may be quite a large region of the chromosome. Therefore, we might wish to narrow this region down by using appropriate crosses between the *sprout* gene and the Noelvirus-resistance gene (typically a backcross between individuals heterozygous for both mutations and individuals homozygous recessive for both mutations) to determine a more precise, and therefore narrower region within which the Noelvirus-resistance gene lies. We could then look for genes within this region that encode proteins that might, when mutant, confer resistance to Noelvirus.

We could then look at the DNA sequences of these genes from phenotypically resistant and sensitive individuals and see whether we could discover a mutation that is always in the resistance alleles, but never in the sensitive alleles.

Question 10

Once we have found the gene that, when mutant, confers resistance to Noelvirus, we could develop a DNA test, possibly a PCR-based DNA test, that allows us to distinguish between turkeys that are resistant and those sensitive to Noelvirus.

We could then isolate the sensitive birds from the resistant birds in the population and protect them from infection. Alternatively, we might simply cull the sensitive birds so that the entire population is resistant. However, as the resistant birds carry the same Noelvirus-resistant allele and are therefore all descendants of the birds in which the resistance allele first arose, the resistant population is likely to be relatively inbred, which itself carries risks. To address this, we could genetically modify the sensitive Noelvirus alleles into resistance alleles in sensitive birds that have been carefully chosen to be genetically distinct from their existing resistant cousins.